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Clinical Significance of Synthesized Posterior/Right-Sided Chest Lead Electrocardiograms in Patients with Acute Chest Pain

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Abstract

Background: 12-lead electrocardiograms (ECGs) provide insufficient information for the accurate diagnosis of posterior and/or right ventricular acute myocardial infarction (AMI) in patients with acute chest pain. Posterior chest leads (V7–V9) and/or right-sided precordial leads (V3R–V5R) provide important information from those specific areas, but these additional ECGs are not routinely recorded because of the time-consuming procedure involved. The purpose of the present study was to evaluate a newly developed system to synthesize these 6 additional lead ECGs non-invasively using standard 12-lead ECG information.

Patients and Methods: Thirty patients (25 men, 5 women; mean age: 65 ± 11 years) complaining of acute chest pain were enrolled. Standard 12-lead and V3R, V4R, V5R, V7, V8, V9 lead ECGs were successively recorded and compared with synthesized ECGs mathematically derived from standard 12-lead signals.

Results: The synthesized and actual ECG waveforms were almost identical, and there were significant correlations in ECG variables, including the P, QRS, and T waves (correlation coefficients about total 1-cycle signals: 0.97 in V3R; 0.93 in V4R; 0.88 in V5R; 0.98 in V7; 0.92 in V8; and 0.88 in V9, $p<0.001$). Both in patients with AMI (N=16) and in patients with ST elevation at the extended leads (N=8), significant correlations were also found (correlation coefficients were over 0.88 at all leads, $p<0.001$). The reproducibility of the ST segment was as good as that of the other ECG variables, even in patients with significant ST elevation due to posterior and/or right ventricular AMI.

Conclusion: Synthesized posterior and right-sided precordial lead ECGs appear to be highly reliable and useful in the rapid diagnosis of AMI, especially in the early detection of posterior and/or right ventricular involvement, thereby alleviating patient distress.
(J Nippon Med Sch 2011; 78: 22–29)

Key words: synthesized electrocardiogram, posterior chest leads, right-sided chest leads, acute myocardial infarction

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Introduction

The accurate and rapid interpretation of electrocardiographic changes is critical in the diagnosis of acute myocardial infarction (AMI) and in the selection of appropriate treatment in patients with chest pain. Although 12-lead electrocardiograms (ECGs) are widely used for this purpose, they provide insufficient information for accurate diagnosis of posterior and/or right ventricular AMI. Posterior chest leads (V7–V9) and/or right-sided precordial leads (V3R–V5R) provide important information from those specific areas and can reveal significant changes when AMI occurs in the posterior wall or right ventricle[1,2]. However, these special lead ECGs are not recorded routinely with conventional ECG machines, because mounting and maintaining electrodes on the surface of the posterior and right-sided anterior chest wall are inconvenient and time-consuming in busy clinical settings requiring urgent decision-making. If electrocardiographic information from these specific areas could be obtained without the necessity of manipulating electrodes on the body surface, it would save much time in reaching a differential diagnosis for patients with chest pain, resulting in better prognoses.

Wei recently designed a theoretical system that allows ECGs corresponding to extended posterior and right-sided precordial leads to be synthesized from 12-lead ECG information[3]. The theoretical basis for this system can be understood from a well-known lead theory that is widely applied in clinical practice. According to this theory, the electrocardiographic potential measured at any location on the body surface is explained as the projection of the equivalent heart source (heart vector) onto a lead vector relative to the electrode position. Eight of the leads for 12-lead ECG (I, II, and V1–V6) were used to estimate the heart vector as an electric source of the patient. Then a transfer matrix, which represents the inherent relation among body surface ECG potentials, was derived on the basis of the least squares method. Assuming fixed conductance around the heart, we calculated the extended lead ECGs with a linear combination of 8 measured ECG signals on the basis of information redundancy in the 12-lead ECG system.

The purposes of the present study were: 1) to evaluate consistency between the synthesized additional lead ECGs obtained via Wei's system and recorded ECGs from those respective areas in actual patients; and 2) to verify the reproducibility of ST levels on the synthesized ECGs when significant ST deviations occur in association with ischemic heart diseases.

Materials and Methods

1. Subjects

Thirty consecutive patients (25 men, 5 women; mean age: 65 ± 11 years) admitted to the Coronary Care Unit (CCU) of Nippon Medical School Hospital complaining of acute chest pain were enrolled in this study. Critically ill patients, including those in shock state or with intolerable severe pain were excluded, as were those who were unable to keep still for ECG recordings. Standard 12-lead ECGs and additional ECGs with leads V3R, V4R, V5R, V7, V8, and V9 were recorded 1 to 3 times depending on the patients' condition on admission and during hospitalization with a Digital Electrocardiograph 1550 (Nihon Kohden, Co. Ltd., Tokyo, Japan). Patients were routinely treated for their respective conditions during the study.

Informed consent was obtained from all patients. The study protocol was approved by the Ethics Committee of Nippon Medical School Hospital.

2. Data Acquisition and Processing for Synthesized ECG

All digital data derived from standard 12-lead ECG signals were processed instantaneously at a sampling rate of 1 kHz. Simulated V3R, V4R, V5R, V7, V8, V9 ECG wave forms were then quickly synthesized with computer software designed by Wei[3,4]. Mathematical differences in the digitalized data between the actual and synthesized signals were calculated point by point with a spreadsheet software program (Excel, Microsoft Corp., Redmond, WA, USA).

3. Measurement and Comparison of ECG Parameters

All ECG variables, including P width, P amplitude, QRS width, QRS amplitude, T amplitude and QT interval, were automatically calculated. These variables were individually compared, and the correlation coefficients between the actual recordings and the synthesized signals from the 6 additional leads were calculated. Then, T-wave morphology was evaluated as 5 patterns: positive deflection only (+), negative deflection only (−), positive and negative deflection (+/−), negative and positive deflection (−/+), and almost flat with no deflection (0). Coincident rates of patterns between the actual and synthesized signals at each lead were derived. In addition, representative 1-beat digitalized data from the onset of the P wave to the end of the T wave (1-cycle total ECG signals comprised serial data of amplitude at each 1-millisecond point) were prepared with every recording of both the actual and synthesized ECGs. The number of digits has depended on the interval from the onset of the P wave to the end of the T wave of each ECG. Then, a correlation coefficient between 2 ECGs was obtained and the mean ± standard deviation of correlation coefficients was calculated at each lead of the evaluated ECGs. In addition to the usual ECG variables, ST deviations (either elevation or depression from the baseline) were evaluated at 3 points (ST-J, ST-M and ST-E) in each recording, as illustrated in **Figure 1**. Absolute differences between the actual and synthesized ECGs at these points were also derived. The intervals between the 3 points were set at 1/16 of the RR interval to avoid any possible influence of sudden changes in T-wave morphology.

4. Statistical Analysis

Correlation coefficients of each ECG variable between recorded and synthesized ECGs were calculated. Because a correlation coefficient about 1-cycle total ECG signals was derived in each ECG, mean ± standard deviations were calculated in each additional leads of evaluated ECGs. Linear regression analysis was used to evaluate the consistency of the above-mentioned ECG variables and waveforms between the recorded and synthesized ECGs. Correlations with $R>0.6$ and $P<0.05$ were considered significant. The absolute differences (in millivolts) are expressed as mean ± standard deviations and were evaluated by paired t-tests; $P<0.05$ was considered statistically significant.

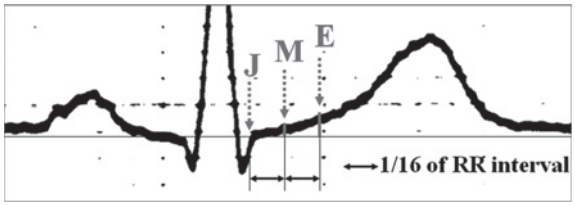

Fig. 1 Measured points of ST deviation from the baseline

Results

Diagnosis of Acute Myocardial Infarction

As shown in **Table 1**, 16 patients with AMI, 6 with congestive heart failure, 3 with aortic diseases, and 1 each with pericarditis, chest pain syndrome, pulmonary thromboembolism, takotsubo cardiomyopathy, and paroxysmal atrial fibrillation were included in this study. Final diagnoses of AMI were made in 16 of the 30 patients with chest pain on the basis of critical elevation of creatine kinase and the MB isozyme of creatine kinase, the presence of Troponin T or heart-type fatty acid-binding protein, and regional wall-motion abnormalities on echocardiograms, in addition to the typical electrocardiographic changes.

The infarcted areas were identified as the right ventricular wall in 3 of the 16 patients and/or the posterior wall in 5 patients on the basis of significant ST elevations actually recorded at the V3R–V5R leads and V7–V9 leads, respectively. Wall-motion abnormalities detected on echocardiograms and perfusion defects on myocardial scintigrams were used as referential data for differential diagnosis when needed.

Correlation between Actual and Synthesized Electrocardiograms

The synthesized signals had almost identical

Table 1 Patient characteristics and final diagnosis

Patient	Age	Sex	Final Diagnosis of Chest Pain	ST elevation
1	74	F	AMI, non-Q anteroseptal	
2	54	M	AMI, inferoposterior	V7–V9
3	60	M	Chest pain syndrome with CLBBB	
4	59	M	AMI, posterior, with CRBBB	
5	58	F	Impending MI, anterior	
6	82	F	CHF due to IHD	
7	63	M	AMI, inferior	
8	60	M	AMI, inferoposterior + RV	V3R–V5R
9	76	M	AMI, inferoposterior	V7–V9
10	72	M	AAD with CRBBB	
11	64	M	CHF due to IHD	
12	72	M	AAD	
13	61	M	AMI, inferoposterior with vasospasm	V7–V9
14	90	M	CPA, CHF due to IHD with CLBBB	
15	68	M	AMI, anteroseptal	
16	69	M	AMI, inferior + RV	V3R-V5R
17	59	M	PAF	
18	81	M	CHF due to IHD with CRBBB	
19	66	M	CHF due to IHD	
20	59	M	AMI, inferior + RV	V3R–V5R
21	49	M	Acute PTE	
22	35	M	Acute pericarditis	
23	71	F	TAA	
24	69	M	Recent MI, non-Q lateral	
25	67	F	CHF due to IHD with CLBBB	
26	50	M	AMI, non-Q posterior	
27	55	M	AMI, anteroseptolateral	
28	78	M	AMI, inferoposterior	V7–V9
29	44	M	AMI, posterior	V7–V9
30	70	M	takotsubo cardiomyopathy	

waveforms to those of the actually recorded electrocardiograms at all of the extended leads, as shown in the examples in **Figures 2 and 3**: the ECG waveforms showing ST elevation at V3R–V5R in a patient with a right ventricular infarction **(Fig. 2)** and at V7–V9 in a patient with a posterior infarction **(Fig. 3)** were clearly reproduced on the synthesized ECGs. Significant correlations in P width, P amplitude, QRS width, QRS amplitude, T amplitude, and QT interval were found between the recorded and synthesized signals as listed in **Table 2** (all $p < 0.001$). The T-wave morphology was coincident with more than 70% of cases in all additional leads. Also, the correlation of 1-cycle total ECG signals was evaluated in 44 ECGs with right-sided leads and in 52 ECGs with posterior leads. Correlation coefficients of each ECG were 0.9 or more in most cases as shown in **Table 3**. No correlation coefficients of less than 0.7 were found in V3R or V7, and only small numbers of low correlation coefficients were observed in the other leads. In 5 cases, correlation coefficients of less than 0.5 were observed, but in all of these cases the amplitudes of the electrical signals in the corresponding actual recordings were very low and were associated with significant noise.

Both in patients with AMI (N=16) and in patients with ST elevation at the extended leads (N=8), significantly high correlations of 1-cycle total ECG signals were also found, even in the case of abnormal ECG waveforms, between the actual and synthesized ECGs in all of the additional leads (**Table 4**).

Reproducibility of ST Levels

Not only deflections in P, QRS, and T waves, but also ST levels were well reproduced in the

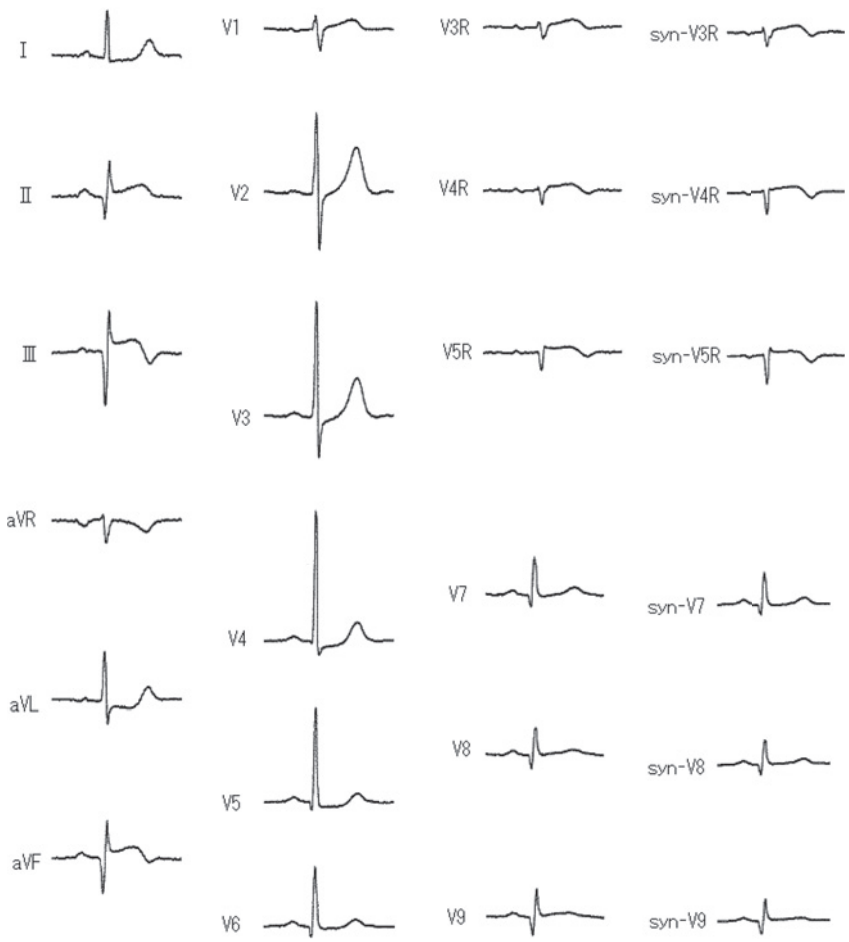

Fig. 2 A case of AMI with ST elevation at V3R–V5R
The patients was a 60-year-old man with inferior and right ventricular AMI.

synthesized signals, even in cases of significant ST elevation or depression, as shown in **Figures 2 and 3**. **Table 5** shows that the differences in ST levels between the recorded and synthesized signals were minimal at all 3 measured points (ST-J, ST-M and ST-E) in the 6 additional leads; absolute differences were less than 0.03 mV at all measured points. The difference tended to be smaller at ST-M than at ST-J and ST-E, but the difference was not statistically significant. Although the difference was trivial at V3R–V5R in cases of right ventricular infarction (N=3, **Table 6a**), it was slightly larger at V7–V9 in cases of posterior infarction (N=5, **Table 6b**), but both the recorded and synthesized signals showed significant ST elevation at the respective leads. Statistical considerations have been omitted because the sample size was small.

Discussion

Convenient and Non-invasive ECG Diagnosis of Posterior/Right Ventricular Infarction

The diagnosis of AMI is usually based on typical symptoms of chest pain with specific changes in 12-lead ECGs and critical elevation of cardiac enzymes. However, it is sometimes difficult to make a definite diagnosis when the infarcted area is localized in the posterior wall or the right ventricle. Although sudden increases in the amplitude of R-waves with ST depression in V1 have often been used as surrogate indicators of posterior infarction[5], the changes in V1 are sometimes very small, resulting in failure to diagnose or misdiagnosis. In most cases of posterior infarction, significant ST elevation appears in leads V7–V9[6–12]. Right ventricular infarction may not be reflected in 12-lead ECGs at all, meaning that

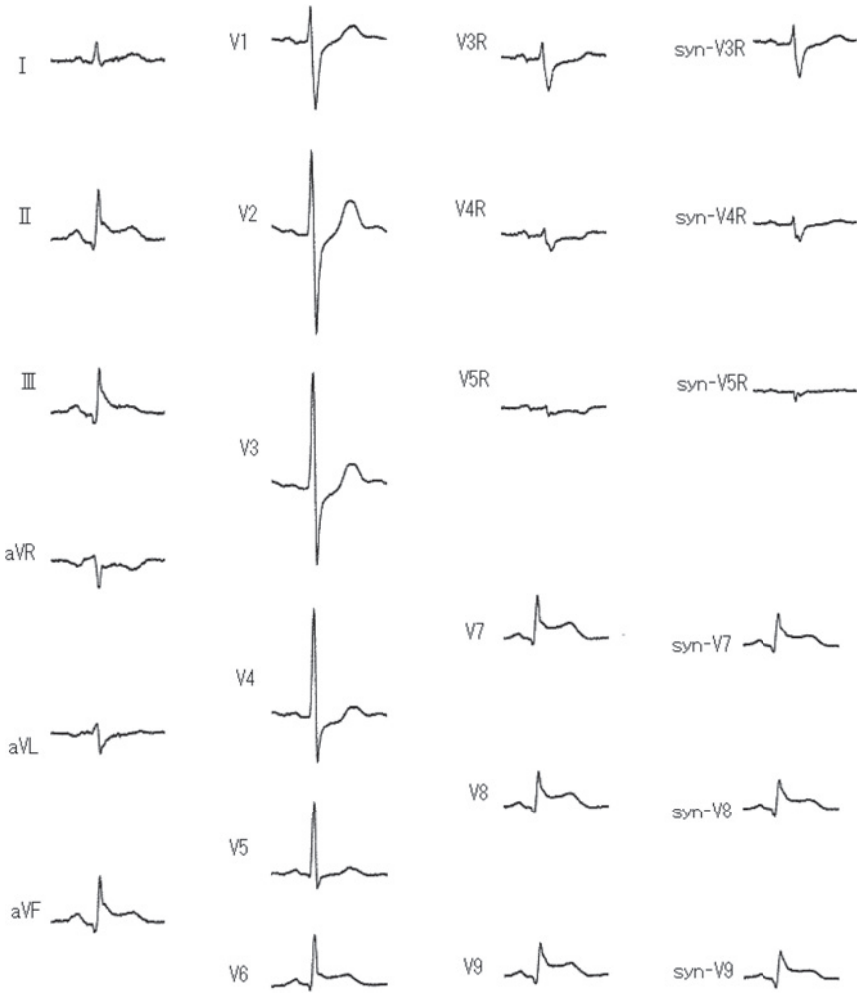

Fig. 3 A case of AMI with ST elevation at V7–V9
The patient was a 61-year-old man with inferoposterior AMI.

Table 2 Correlation coefficients of ECG parameters between actual and synthesized ECGs

	V3R	V4R	V5R	V7	V8	V9
P width	0.97	0.94	0.91	0.98	0.94	0.93
P amplitude	0.88	0.93	0.92	0.94	0.85	0.81
QRS width	0.97	0.91	0.88	0.98	0.91	0.86
QRS amplitude	0.96	0.92	0.89	0.92	0.81	0.81
QT interval	0.93	0.91	0.84	0.93	0.86	0.88
T amplitude	0.96	0.93	0.93	0.89	0.79	0.65
T morphology Coincident rate(%)	86.0	72.1	81.4	82.7	82.7	82.7

N=44 for V3R–V5R, N=52 for V7–V9

ST elevation at V3R–V5R may be the only clear indication of right ventricular infarction[13–15]. However, few commercial electrocardiographs have the capacity to make simultaneous recordings of ECGs from leads other than the standard 12. When AMI of the posterior wall or the right ventricle is suspected in clinical settings, readings from several additional leads, including V3R, V4R, V5R, V7, V8, and V9, should be recorded after 12-lead ECG recordings have been done, whether typical ST changes are detected or not. However, this is time-consuming and inconvenient when urgent treatment of AMI is

required. If these additional lead ECG readings could be derived quickly and non-invasively from the standard 12-lead information, it would benefit both physicians and patients greatly and quite possibly lead to better outcomes. In the present study, we obtained good correlations between actual and synthesized ECGs at the additional leads, and high reproducibility of elevated ST level findings. Thus, we believe that our system meets the needs of both physicians and patients and can be widely used in clinical settings for differential diagnosis in patients with chest pain.

Table 3 Distribution of correlation coefficients of 1-cycle ECG signals between actual and synthesized signals

R=	V3R	V4R	V5R	V7	V8	V9
>0	0	0	0	0	0	1
0–0.19	0	0	1	0	1	0
0.2–0.49	0	0	1	0	1	0
0.5–0.69	0	1	2	0	0	2
0.7–0.79	1	2	2	0	4	7
0.8–0.89	2	7	7	2	3	5
0.9–1.0	41	34	31	50	43	37

Correlation and Reproducibility of Synthesized ECGs with Actually Recorded ECGs

The waveforms of the mathematically-derived signals on the extended posterior and right-sided precordial leads using Wei's method were almost identical to the actual ECGs recorded in patients with chest pain. Good correlations were obtained between actual and synthesized ECGs at all additional leads, not only in cases of chest pain, but also in cases of AMI and ST elevation. However, the correlation coefficients tended to become smaller in proportion to the distance of the lead from the heart, i.e., from V3R to V5R, and from V7 to V9. This phenomenon might be explained by the fact that greater distance from the heart results in lower amplitudes on ECGs. We suspect that the correlation coefficient depends partly on the amplitude of the total signals.

In most of the patients with significant ST elevations, the ST changes were also reproduced

Table 4 Mean correlation coefficients of 1-cycle total ECG signals in patients with AMI or ST elevation

	V3R	V4R	V5R	V7	V8	V9
All patients (N=30)	0.97 ± 0.06	0.93 ± 0.08	0.88 ± 0.18	0.98 ± 0.02	0.92 ± 0.18	0.89 ± 0.18
AMI (N=16)	0.96 ± 0.07	0.91 ± 0.07	0.84 ± 0.21	0.98 ± 0.03	0.90 ± 0.22	0.88 ± 0.19
ST elevated (N=8)	0.97 ± 0.02	0.91 ± 0.04	0.87 ± 0.14	0.98 ± 0.04	0.97 ± 0.06	0.94 ± 0.06

Mean ± SD

Table 5 Absolute differences in ST levels between actual and synthesized ECGs

	V3R	V4R	V5R	V7	V8	V9
ST-J	0.019 ± 0.017	0.019 ± 0.018	0.016 ± 0.018	0.018 ± 0.019	0.025 ± 0.025	0.028 ± 0.025
ST-M	0.018 ± 0.019	0.018 ± 0.021	0.017 ± 0.019	0.016 ± 0.023	0.018 ± 0.026	0.022 ± 0.029
ST-E	0.023 ± 0.024	0.025 ± 0.025	0.021 ± 0.022	0.020 ± 0.031	0.022 ± 0.037	0.027 ± 0.042

Mean ± SD (mV)

Table 6a Absolute differences in ST levels in cases of RV infarction

	V3R	V4R	V5R
ST-J	0.003 ± 0.004	0.003 ± 0.004	0.010 ± 0.007
ST-M	0.013 ± 0.018	0.020 ± 0.021	0.018 ± 0.011
ST-E	0.020 ± 0.014	0.025 ± 0.021	0.025 ± 0.007

Mean ± SD (mV)

Table 6b Absolute differences in ST levels in cases of posterior infarction

	V7	V8	V9
ST-J	0.033 ± 0.029	0.033 ± 0.039	0.040 ± 0.039
ST-M	0.031 ± 0.039	0.033 ± 0.046	0.038 ± 0.050
ST-E	0.039 ± 0.056	0.044 ± 0.066	0.050 ± 0.076

Mean ± SD (mV)

accurately in the synthesized ECGs. The absolute differences in the ST levels were less than 0.03 mV between the recorded and synthesized ECGs, which we considered insignificant for the purpose of differential diagnosis of AMI. Although the difference tended to be slightly smaller at ST-M than at ST-J and ST-E, the difference was not statistically significant. We think that ST-M might be more stable than the other points, because the influences of changes in the depth and duration of S waves and of changes in T-wave morphology are smaller at ST-M. In any event, these results suggest that posterior and/or right ventricular infarction can be diagnosed without actual ECG recordings from additional posterior or right-sided chest leads in clinical settings.

In conclusion, we believe that the synthesized 18-lead electrocardiogram designed by Wei is very useful in the rapid diagnosis of AMI, especially in the early detection of posterior and/or right ventricular involvement, thereby alleviating patient distress.

Limitations

The number of patients included in the study was small, because most patients admitted to the CCU complaining of severe chest pain were excluded as subjects owing to the critical nature of their conditions. Another limitation is that because of the small number of patients, correlations between the actual and synthesized ECGs could not be evaluated in terms of the extent or area of infarction. A larger study population with infarctions of various sizes in different areas will be needed for a more reliable analysis of ST level reproducibility.

Acknowledgement: The authors wish to express their gratitude to the CCU staff doctors for their great assistance in collecting ECG data.

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(Received, July 26, 2010)
(Accepted, November 1, 2010)